Acute myocardial involvement after heroin inhalation

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ABSTRACT

Amongst the illicit drugs cocaine, amphetamines and cannabis have been studied and documented well to cause myocardial infarction by different mechanisms but there is very sparse data available on myocardial involvement after heroin abuse. We report a young man who developed acute myocardial injury after heroin inhalation and alcohol binge drinking. Heroin induced cardio toxic effect and vasospasm compounded by alcohol were suspected to be the cause of this.

Key words: Acute myocardial infarction, binge drinking, heroin abuse, myocardial infarction in young

INTRODUCTION

The complications related to drug abuse such as myocardial insufficiency, myocardial infarction, endocarditis, myocarditis, aortic dissection, neurologic damage, ischemic colitis and renal failure are known, but have been reported very rarely.

Amongst the illicit drugs, cocaine, amphetamines and cannabis have been studied and documented well to cause myocardial infarction by different mechanisms. They constitute an important cause of coronary artery disease in young patients. Drug overdose is a major cause of premature death and morbidity among heroin abusers. There is very sparse data available on myocardial involvement after heroin abuse.[1-3] Here, we report a young male who suffered from acute myocardial injury after heroin inhalation.

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CASE REPORT

A 28-year-old young male shopkeeper was brought to the emergency with complaints of chest discomfort, cold sweating and severe generalized weakness. He had no history of valvular or congenital heart disease. There was no history of fever, headache, any flu-like symptoms, systemic illness or drug allergy. His parents were alive and there was no history of diabetes mellitus, coronary artery disease, hypertension or cerebrovascular disease in first degree relatives. He was not a cigarette smoker but his parents and patient himself gave history of heroin inhalation (smack) for last five years. He was married and having strained relations with his wife. As per history last heroin inhalation was done approximately 12 h before admission along with alcohol binge drinking. On general physical examination he was thin built, looked drowsy but arousable. His vital signs were pulse 64/min, blood pressure 70/50 mmHg, respiratory rate 24/min and temperature was 98.6°F along with cold extremities. His both the pupils were small and pinpoint. No other abnormal finding was observed in general examination. The systemic examination was also normal except the chest auscultation which revealed basal crepitations. Laboratory data showed mild leukocytosis with total leukocyte count of 14,200/cmm, hemoglobin 14.6 gm%, platelet count 2.6 lacs/cmm. Biochemical investigations including urea, creatinine, total protein, albumin, liver

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transaminases, glucose and fasting lipid profile were within normal limits. Chest skiagram showed mild pulmonary congestion but no cardiomegaly. Electrocardiogram revealed ST segment elevation V_1-V_4. Creatine phophokinase-MB was raised 91U/L (normal value<20U/L) Total CPK was 456 U/L(normal being <200) and toponin-I was 3.02 ng/ml (normal value <0.01 ng/ml). In 2-D echocardiography hypo kinesis of left ventricular anterior wall was noticed while left ventricular systolic function and ejection fraction was within normal limits (64%).On the basis of history, clinical examination and investigations, diagnosis of acute coronary ischemic event involving left ventricular wall was made. Since the patient had hypotension at presentation, he was treated with aspirin 325 mg and Clopidogrel 300 mg and vasopressors, but nitroglycerine could not be given to him. The patient did not receive thrombolytic therapy, because of the pattern of electrocardiographic changes and time interval between the onset of chest tightness and hospitalization was more than 12 h. Therefore,he was administered only subcutaneous low molecular weight heparin Enoxaparin. He responded to treatment, got relief in the chest discomfort and was discharged after seven days. At the time of discharge, minimal ST elevation persisted in electrocardiogram which became normal after a week during follow up along with the echocardiography. Coronary angiography which was done one month later did not reveal any significant luminal stenosis.

DISCUSSION

The patient was diagnosed to have acute myocardial involvement on the basis of symptoms of chest tightness, cold sweating and hypotension. His investigations that included electrocardiogram, echocardiography, CPK-MB and Troponin-I and also favored the diagnosis of acute myocardial injury. He had bilateral constricted pupils with excessive salivation, basal crepitations in the chest and relative bradycardia despite hypotension. There was a definite history of heroin inhalation and binge drinking in the night prior to admission. No family history of premature heart disease or any conventional risk factors of cardiovascular disease were present in the patient.

Our patient presented with symptom of chest tightness, which was not localized, had no postural variation or relation with respiration. So it was not probably pericarditis. He possibly had acute myocardial ischemia which is being supported by elevated cardiac biomarkers, electrocardiographic changes and echocardiography. Myocarditis can also masquerade as myocardial ischemia sometimes because of ST-T changes, cardiac enzyme elevation and wall motion abnormalities which can be observed with myocarditis. Myocarditis was a possibility in our patient that could not be excluded. During the hospitalization we could not subject the patient for either coronary angiography or myocardial scintigraphy to confirm the infarct or any occlusive lesion in the coronaries.

Heroin (diacetylmorphine) is rapidly hydrolyzed to 6-monoacetylmorphine which in turn is hydrolyzed to morphine. Though heroin-induced myocardial infarction has been seldom reported,[1,2] the underlying mechanisms have been still unclear. Heroin may have systemic effect or may be directly cardio toxic. Heroin associated rhabdomyolysis with cardiac involvement have been described earlier. Melandri *et al*.[3] reported a case of heroin intoxication followed by rhabdomyolysis associated with myocardial injury, with symptoms, laboratory findings, ECG and echocardiography features of non-Q wave infarction. However, a 201 Thalium myocardial scintigraphy, performed after patient was discharged, did not show any abnormality. This shows that heroin probably has a direct myotoxic effect on both myocardium and skeletal muscle. There is a possibility that hypoxia, acidosis, vasoconstrictive substances released by muscle necrosis, or hypersensitivity reactions associated with heroin or some of its adulterants are involved in myocardial injury.

Sztajzel *et al*.[1] reported a young woman with acute myocardial infarction after heroin abuse; they had claimed that heroin might have a direct toxic effect on the coronary arteries and can cause coronary occlusion by provoking vasospasm or inflammation. There has been suggestion that heroin acts directly on vasomotor centre to increase parasympathetic activity and reduces sympathetic tone, which leads to vasodilatation and stimulates histamine release from mast cells. These effects are responsible for bradycardia and hypotension[4] which was present in our patient too. There was history of binge drinking present in our patient which has been associated with development of acute myocardial infarction.[5] Binge drinking is particularly dangerous form of alcohol consumption. Men who consume five drinks (50 g) or more and women who consume four drinks (40 g) or more in single sitting are known as binge drinkers. It is well known that ethanol induces concentration dependent vasospasm in coronary arteries and coronary vasoconstriction may itself damage the endothelium thereby increasing the likelihood of platelet adhesion and thrombus formation.

In conclusion, a 28-year-old young male developed acute myocardial injury after heroin inhalation and binge drinking while he had no other conventional cardiovascular risk factors. The cause might be heroin-induced cardio toxic effect or vasospasm compounded by the presence of binge drinking.

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